

**Citation:**

Halton TL, Willett WC, Liu S, Manson JE, Stampfer MJ, Hu FB. Potato and french fry consumption and risk of type 2 diabetes in women. *Am J Clin Nutr*. 2006;83(2):284-90.

**PubMed ID:** [16469985](#)

**Study Design:**

Prospective Cohort Study

**Class:**

B - [Click here](#) for explanation of classification scheme.

**Research Design and Implementation Rating:**

POSITIVE: See Research Design and Implementation Criteria Checklist below.

**Research Purpose:**

The objective was to examine prospectively the relation between potato consumption and the risk of type 2 diabetes.

**Inclusion Criteria:**

- Inclusion in the The Nurses' Health Study

**Exclusion Criteria:**

- Women at baseline who left >/- 10 food items blank on the semiquantitative FFQ.
- Implausibly high (>3500 kcal) or low (< 500 kcal) energy intakes on the semiquantitative FFQ.
- History of diabetes, cancer (not including nonmelanoma skin cancer), or cardiovascular disease at baseline.

**Description of Study Protocol:****Recruitment**

- The Nurses' Health Study
- Mailed questionnaires to female registered nurses aged 30 - 55y.

**Design:** Prospective cohort study

**Blinding used (if applicable)**

- Implied for analysis of dietary data

**Intervention (if applicable):** not applicable

## Statistical Analysis

- Each participant contributed follow-up time from the date of returning the 1980 baseline questionnaire to the date of diagnosis of type 2 diabetes, 1 June 2000, or death.
- Women were excluded from additional followup once they were diagnose with diabetes.
- The participants were divided into 5 categories (quintiles) according to the frequency of potato consumption.
- To represent long-term intake and to reduce measurement error, the cumulative frequency of consumption was calculated.
- Quintiles of cumulative french fry consumption were also created.
- Incidence rates for type 2 diabetes were calculated by dividing cases by the person-years of follow-up for each quintile of potato intake.
- Relative risks (RRs) of type 2 diabetes were calculated by dividing the rate of occurrence of diabetes in each quintile by the rate in the first (lowest) quintile.
- Cox proportional hazards models were used to adjust for potentially confounding variables, which included BMI, family history of diabetes, smoking, postmenopausal hormone use, and physical activity. The investigators additionally adjusted for dietary variables, including *trans* fat, the ratio of polyunsaturated fat to saturated fat, cereal fiber, and total calories.
- Potatoes and whole grains were considered as continuous variables in the same model.
- The difference in the coefficients from this multivariate model was used to estimate the RR and 95% CI for substituting 1 serving potatoes/d for 1 serving whole grains and refined grains/d.
- All P values were two-sided. Tests for trend were examined by using the median value for each category of potato consumption, which was analyzed as a continuous variable in the regression models.
- All statistical analyses were performed with SAS version 8.2 software.

## Data Collection Summary:

### Timing of Measurements

- Diet was assessed in 1980, 1984, 1986, 1990, 1994, and 1998.
- Since 1976, information on disease status as well as lifestyle factors was collected every 2 years.
- The participants were followed for 20 years (1980 - 2000).

### Dependent Variables

- Incident/relative risk of type 2 diabetes mellitus based on self-report of diagnosis and/or symptoms

### Independent Variables

- Potato consumption
- French fry consumption

### Control Variables

- BMI
- Family history of diabetes
- Smoking
- Postmenopausal hormone use

- Physical activity
- Additionally adjusted for dietary variables, including trans fat, the ratio of polyunsaturated fat to saturated fat, cereal fiber, and total calories.

## **Description of Actual Data Sample:**

### **Initial N:**

- In 1976, 121,799 female registered nurses completed a mailed questionnaire.

### **Attrition (final N):**

- After exclusions, 84,555 women remained in this investigation.

**Age:** 30 - 55 y at baseline

**Ethnicity:** 98% were white which reflected the ethnic composition of US registered nurses at the time.

**Other relevant demographics:** none given

### **Anthropometrics**

- BMI: ranged from 25.0 to 26.0

### **Location:**

- Participants assumed to be throughout US.
- Data analysis at Brigham and Women's Hospital, Boston, MA.

## **Summary of Results:**

### **Key Findings**

- At baseline in 1980, potato consumption ranged from a median of 0.07 serving/d in the first quintile to 0.79 serving/d in the highest quintile.
- French fry intake was considerably less; the median for the lowest quintile was zero servings, whereas the median in the highest quintile was just 0.14 serving/d.
- The amount of consumption was similar to that of the general US population during the years 1980 - 2000.
- In additional analyses that used potato consumption as a continuous variable, the multivariate RR of type 2 diabetes for consuming 1 serving potatoes/d [237 mL (1 cup) mashed or 1 baked] was 1.18 (95% CI: 1.03, 1.35).
- The multivariate RR for 2 [113 g (4 oz)] servings french fries/wk was 1.16 (95% CI: 1.05, 1.29).
- The RR of substituting 1 serving potatoes/d for 1 serving whole grains/d was 1.30 (95% CI: 1.08, 1.57). The RR of substituting 1 serving potatoes/d for 1 serving refined grains/d was 1.22 (95% CI: 1.01, 1.47).
- The multivariate RR of the type 2 diabetes in a comparison between the highest and lowest quintiles (median=0.14 vs. zero servings per day) of french fry intake was 1.21 (95% CI: 1.09, 1.33; p for trend <0.0001)

### **Other Findings**

- 4496 cases of type 2 diabetes were documented during the 20 year followup.
- Potato and french fry consumption was largely consistent over time (overall mean  $\pm$  SD: 0.32  $\pm$  0.23 servings potatoes/d for potato and 0.07  $\pm$  0.08 servings french fries/d).
- The women who consumed more potatoes tended to have a higher dietary glycemic load and higher intakes of red meat, refined grain, and total calories.
- The women who consumed more french fries tended to have a higher dietary glycemic load and higher intakes of red meat, refined grain and total calories.
- Family history of diabetes, *trans* fat intake, BMI, and physical activity were not significantly different across quintiles for either potato or french fry consumption (as shown in Table 1 of article).
- Potatoes: age-adjusted RR of type 2 diabetes was 1.13 (95% CI: 1.03, 1.25) in a comparison between the women in the fifth quintile and those in the first quintile (P for trend = 0.02).
- In multivariate models, the addition of BMI as a categorical variable slightly increased the RR to 1.18 (95% CI: 1.07, 1.30; P for trend = 0.0003).
- Potential confounding variables did appreciably change the RR.
- Stratified analyses demonstrated the association between potato consumption and diabetes was statistically significant in obese women but not in nonobese women (P for interaction = 0.01). This was not observed for french fry consumption.
- No significant interaction was observed between the consumption of potatoes or french fries and physical activity or family history of type 2 diabetes.
- Red meat was significantly correlated with consumption of potatoes ( $r = 0.24$ ,  $P < 0.0001$ ) and french fries ( $r = 0.29$ ,  $P < 0.0001$ ). Inclusion of red meat consumption into the multivariate models slightly attenuated the RR of type 2 diabetes.
- To examine whether the associations between potatoes and french fry intake were mediated through a higher glycemic load, glycemic load was added to the multivariate models. After adjustment for glycemic load, the RRs in a comparison of extreme quintiles were 1.06 (95% CI: 0.95, 1.18; P for trend = 0.24) for potatoes and 1.15 (95% CI: 1.04, 1.27; P for trend = 0.005) for french fries.

### Author Conclusion:

Higher consumption of potatoes and french fries was associated with a modestly increased risk of type 2 diabetes in this large prospective cohort of women. The increased risk was more pronounced when potatoes replaced whole-grain products in the diet. The association was independent of known risk factors for type 2 diabetes. These data support a potential benefit from limiting consumption of these foods in reducing the risk of type 2 diabetes. Substitution of these sources of carbohydrates with lower glycemic, high-fiber forms of carbohydrates such as whole grains should be encouraged.

### Reviewer Comments:

*As mentioned by the authors, the homogeneity of the population (mostly white women with some college education) limits the ability to evaluate the association between potatoes and the risk of type 2 diabetes in women of other racial and educational backgrounds. Diagnosis of diabetes based on self-report.*

## Research Design and Implementation Criteria Checklist: Primary Research

### Relevance Questions

- |    |   |     |
|----|---|-----|
| 1. | Would implementing the studied intervention or procedure (if found successful) result in improved outcomes for the patients/clients/population group? (Not Applicable for some epidemiological studies) | Yes |
| 2. | Did the authors study an outcome (dependent variable) or topic that the patients/clients/population group would care about?   | Yes |
| 3. | Is the focus of the intervention or procedure (independent variable) or topic of study a common issue of concern to nutrition or dietetics practice?  | Yes |
| 4. | Is the intervention or procedure feasible? (NA for some epidemiological studies)  | Yes |

### Validity Questions

- |      |   |     |
|------|---|-----|
| 1.   | <b>Was the research question clearly stated?</b>  | Yes |
| 1.1. | Was (were) the specific intervention(s) or procedure(s) [independent variable(s)] identified?   | Yes |
| 1.2. | Was (were) the outcome(s) [dependent variable(s)] clearly indicated?  | Yes |
| 1.3. | Were the target population and setting specified?   | Yes |
| 2.   | <b>Was the selection of study subjects/patients free from bias?</b>   | Yes |
| 2.1. | Were inclusion/exclusion criteria specified (e.g., risk, point in disease progression, diagnostic or prognosis criteria), and with sufficient detail and without omitting criteria critical to the study? | Yes |
| 2.2. | Were criteria applied equally to all study groups?  | N/A |
| 2.3. | Were health, demographics, and other characteristics of subjects described?   | Yes |
| 2.4. | Were the subjects/patients a representative sample of the relevant population?  | Yes |
| 3.   | <b>Were study groups comparable?</b>  | Yes |
| 3.1. | Was the method of assigning subjects/patients to groups described and unbiased? (Method of randomization identified if RCT)   | Yes |
| 3.2. | Were distribution of disease status, prognostic factors, and other factors (e.g., demographics) similar across study groups at baseline?  | Yes |
| 3.3. | Were concurrent controls used? (Concurrent preferred over historical controls.)   | Yes |

3.4.	If cohort study or cross-sectional study, were groups comparable on important confounding factors and/or were preexisting differences accounted for by using appropriate adjustments in statistical analysis?	Yes
3.5.	If case control or cross-sectional study, were potential confounding factors comparable for cases and controls? (If case series or trial with subjects serving as own control, this criterion is not applicable. Criterion may not be applicable in some cross-sectional studies.)	N/A
3.6.	If diagnostic test, was there an independent blind comparison with an appropriate reference standard (e.g., "gold standard")?	N/A
<b>4.</b>	<b>Was method of handling withdrawals described?</b>	Yes
4.1.	Were follow-up methods described and the same for all groups?	Yes
4.2.	Was the number, characteristics of withdrawals (i.e., dropouts, lost to follow up, attrition rate) and/or response rate (cross-sectional studies) described for each group? (Follow up goal for a strong study is 80%.)	Yes
4.3.	Were all enrolled subjects/patients (in the original sample) accounted for?	Yes
4.4.	Were reasons for withdrawals similar across groups?	N/A
4.5.	If diagnostic test, was decision to perform reference test not dependent on results of test under study?	N/A
<b>5.</b>	<b>Was blinding used to prevent introduction of bias?</b>	Yes
5.1.	In intervention study, were subjects, clinicians/practitioners, and investigators blinded to treatment group, as appropriate?	N/A
5.2.	Were data collectors blinded for outcomes assessment? (If outcome is measured using an objective test, such as a lab value, this criterion is assumed to be met.)	Yes
5.3.	In cohort study or cross-sectional study, were measurements of outcomes and risk factors blinded?	N/A
5.4.	In case control study, was case definition explicit and case ascertainment not influenced by exposure status?	N/A
5.5.	In diagnostic study, were test results blinded to patient history and other test results?	N/A
<b>6.</b>	<b>Were intervention/therapeutic regimens/exposure factor or procedure and any comparison(s) described in detail? Were intervening factors described?</b>	Yes
6.1.	In RCT or other intervention trial, were protocols described for all regimens studied?	N/A
6.2.	In observational study, were interventions, study settings, and clinicians/provider described?	Yes

6.3.	Was the intensity and duration of the intervention or exposure factor sufficient to produce a meaningful effect?	Yes
6.4.	Was the amount of exposure and, if relevant, subject/patient compliance measured?	Yes
6.5.	Were co-interventions (e.g., ancillary treatments, other therapies) described?	N/A
6.6.	Were extra or unplanned treatments described?	N/A
6.7.	Was the information for 6.4, 6.5, and 6.6 assessed the same way for all groups?	Yes
6.8.	In diagnostic study, were details of test administration and replication sufficient?	N/A
<b>7.</b>	<b>Were outcomes clearly defined and the measurements valid and reliable?</b>	<b>Yes</b>
7.1.	Were primary and secondary endpoints described and relevant to the question?	Yes
7.2.	Were nutrition measures appropriate to question and outcomes of concern?	Yes
7.3.	Was the period of follow-up long enough for important outcome(s) to occur?	Yes
7.4.	Were the observations and measurements based on standard, valid, and reliable data collection instruments/tests/procedures?	Yes
7.5.	Was the measurement of effect at an appropriate level of precision?	Yes
7.6.	Were other factors accounted for (measured) that could affect outcomes?	Yes
7.7.	Were the measurements conducted consistently across groups?	N/A
<b>8.</b>	<b>Was the statistical analysis appropriate for the study design and type of outcome indicators?</b>	<b>Yes</b>
8.1.	Were statistical analyses adequately described and the results reported appropriately?	Yes
8.2.	Were correct statistical tests used and assumptions of test not violated?	Yes
8.3.	Were statistics reported with levels of significance and/or confidence intervals?	Yes
8.4.	Was "intent to treat" analysis of outcomes done (and as appropriate, was there an analysis of outcomes for those maximally exposed or a dose-response analysis)?	N/A
8.5.	Were adequate adjustments made for effects of confounding factors that might have affected the outcomes (e.g., multivariate analyses)?	Yes
8.6.	Was clinical significance as well as statistical significance reported?	Yes



8.7.	If negative findings, was a power calculation reported to address type 2 error?	N/A
<b>9.</b>	<b>Are conclusions supported by results with biases and limitations taken into consideration?</b>	<b>Yes</b>
9.1.	Is there a discussion of findings?	Yes
9.2.	Are biases and study limitations identified and discussed?	Yes
<b>10.</b>	<b>Is bias due to study's funding or sponsorship unlikely?</b>	<b>Yes</b>
10.1.	Were sources of funding and investigators' affiliations described?	Yes
10.2.	Was the study free from apparent conflict of interest?	Yes

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